AMENDMENTS TO THE CLAIMS

This listing will replace all prior claim listings in the application:

1-13. (CANCELED)

14. (PREVIOUSLY PRESENTED) The method of claim 65 wherein occurrence of an individual disorder associated polymorphism is assessed by

contacting a nucleic acid derived from the human's genome with a first oligonucleotide that anneals with higher stringency with the disorder associated polymorphism than with a corresponding non-disorder associated polymorphism, and

assessing annealing of the first oligonucleotide and the nucleic acid,

whereby annealing of the first oligonucleotide and the nucleic acid indicates that the human's genome comprises the disorder associated polymorphism.

15. (PREVIOUSLY PRESENTED) The method of claim 65 wherein the first oligonucleotide is attached to a support.

16. (PREVIOUSLY PRESENTED) The method of claim 15 wherein the support has a plurality of different first oligonucleotides attached thereto.

17. (PREVIOUSLY PRESENTED) The method of claim 16 wherein the support has attached thereto at least two first oligonucleotides that anneal with higher stringency with the disorder associated polymorphisms than with the corresponding non-disorder associated polymorphisms.

18-19. (CANCELED)

20. (PREVIOUSLY PRESENTED) The method of claim 14 wherein the first oligonucleotide is a molecular beacon oligonucleotide.

21. (PREVIOUSLY PRESENTED) The method of claim 14 wherein occurrence of an individual disorder associated polymorphism is further assessed by

contacting the nucleic acid with a second oligonucleotide that anneals with higher stringency with a non-disorder associated polymorphism than with the corresponding disorder associated polymorphism, and

assessing annealing of the second oligonucleotide and the nucleic acid,

whereby annealing of the second oligonucleotide and the nucleic acid indicates that the human's genome does not comprise the disorder associated polymorphism.

22. (PREVIOUSLY PRESENTED) The method of claim 21 wherein the second oligonucleotide is attached to a support.

23 (PREVIOUSLY PRESENTED) The method of claim 22 wherein the first and second oligonucleotides are attached to the same support.

24. (PREVIOUSLY PRESENTED) The method of claim 21 wherein the second oligonucleotide is a molecular beacon oligonucleotide.

25. (PREVIOUSLY PRESENTED) The method of claim 24 wherein the first and second oligonucleotides are spectrally distinct molecular beacon oligonucleotides.

26. (CURRENTLY AMENDED) The method of claim 65 further comprising calculating the susceptibility value by summing, for each of the gene encoding a vitamin D receptor and the gene encoding interleukin-6 in which the disorder associated polymorphism occurs in the human's genome, the product of a constant and a correlation factor, wherein the correlation factor represents the fraction of humans heterozygous or homozygous for the disorder associated polymorphism who exhibit the corresponding disorder, whereby the susceptibility ~~score~~ value represents the relative susceptibility of the human to an undesirable bone density condition.

27. (PREVIOUSLY PRESENTED) The method of claim 26 wherein the same constant is used for each selected gene.

28-29. (CANCELED)

30. (CURRENTLY AMENDED) The method of claim [[1]] 14 wherein at least one of the disorder associated polymorphisms is a single nucleotide polymorphism (SNP).

31. (PREVIOUSLY PRESENTED) The method of claim 30 wherein occurrence of a SNP is assessed by annealing a nucleic acid derived from the human's genome with a primer that is complementary to the region adjacent the SNP on its 3' side, extending the primer using a polymerase in order to add a nucleotide residue complementary to the SNP to the primer, and detecting the identity of the nucleotide residue complementary to the SNP.

32. (PREVIOUSLY PRESENTED) The method of claim 31 wherein the nucleotide residue is a non-extendable residue.

33. (PREVIOUSLY PRESENTED) The method of claim 30 wherein the SNP is selected from the group consisting of

a) occurrence of a thymine residue 8 residues upstream of the normal start codon of the gene encoding vitamin D receptor, whereby the residue is part of an initiation codon and the gene encodes a variant vitamin D receptor comprising three additional amino acids at its amino terminus; and

b) occurrence of a cytosine residue at position -174 of the interleukin 6 gene promoter.

34-62. (CANCELED)

63. (CURRENTLY AMENDED) The method of claim [[1]] 14 wherein the disorder associated polymorphisms include at least:

a) occurrence of a thymine residue 8 residues upstream of the normal start codon of the gene encoding vitamin D receptor, whereby the residue is part of an initiation codon and the gene encodes a variant vitamin D receptor comprising three additional amino acids at its amino-terminus; and

b) occurrence of a cytosine residue at position -174 of the interleukin 6 gene promoter.

64. (CANCELED)

65. (CURRENTLY AMENDED) A method comprising

assessing a relative degree to which a human is susceptible to an undesirable bone density condition by identifying a polymorphic form identified as associated with ~~any~~ a bone density pathology in each of

a gene encoding a vitamin D receptor present in the human's genome, and

a gene encoding interleukin-6 present in the human's genome,

thereafter calculating a susceptibility value for the condition by either

summing the identified polymorphisms to yield a value for the human, or

assigning a weighting factor to each polymorphism and then summing the weighting factors to yield a value for the human,

wherein a value for the human greater than a value for a control indicates a greater susceptibility to the undesirable bone density condition for the human, and wherein the polymorphic form is a disorder associated polymorphism,

the method hereby assessing the relative degree to which the human is susceptible to the undesirable bone density condition.

66. (PREVIOUSLY PRESENTED) The method of claim 65 wherein the undesirable bone density condition is selected from the group consisting of undesirable bone formation, undesirable bone erosion, or undesirable bone resorption.

67. (PREVIOUSLY PRESENTED) The method of claim 65 where the weighting factor is a score indicating significance of the disorder-associated polymorphism.

68. (PREVIOUSLY PRESENTED) The method of claim 65 wherein the weighting factor is a score correlating polymorphism homozygosity and a bone density associated disorder.

69. (NEW) The method of claim 65 wherein the susceptibility value for the control is zero and represents an absence of a polymorphic form identified as associated with a bone density pathology in each of a gene encoding a vitamin D receptor present in the human's genome and a gene encoding interleukin-6 present in the human's genome.

70. (NEW) The method of claim 65 wherein the polymorphic form is a single nucleotide polymorphism (SNP).

71. (NEW) A method comprising

assessing a relative degree to which a human is susceptible to an undesirable bone density condition by identifying a polymorphic form identified as associated with a bone density pathology in each of

a gene encoding a vitamin D receptor present in the human's genome, and

a gene encoding interleukin-6 (IL-6) present in the human's genome

wherein the polymorphic form is selected from the group consisting of

a) occurrence of a *FokI* polymorphism in the gene encoding vitamin D receptor defined by a C/T nucleotide in exon 2, at the first of two potential translation initiation sites, whereby the residue is part of an initiation codon and the gene encodes a variant vitamin D receptor comprising three additional amino acids at its amino terminus;

b) occurrence of a *BsmI* polymorphism in the gene encoding a vitamin D receptor defined by a T/C change in intron 8;

c) occurrence of a *ApaI* polymorphism in the gene encoding a vitamin D receptor defined by a T/G change in intron 8;

d) occurrence of a *TaqI* polymorphism in the gene encoding a vitamin D receptor defined by a T/C change in exon 9; and

e) occurrence of a polymorphism in the IL-6 gene promoter defined by a G/C change at position -174,

thereafter calculating a susceptibility value for the condition by either

summing the identified polymorphisms to yield a value for the human, or

assigning a weighting factor to each polymorphism and then summing the weighting factors to yield a value for the human,

wherein a value for the human greater than a value for a control indicates a greater susceptibility to the undesirable bone density condition for the human, and wherein the polymorphic form is a disorder associated polymorphism,

the method hereby assessing the relative degree to which the human is susceptible to the undesirable bone density condition.

72. (NEW) The method of claim 71 wherein the susceptibility value for the control is zero and represents an absence of a polymorphic form identified as associated with a bone density pathology in each of a gene encoding a vitamin D receptor present in the human's genome and a gene encoding interleukin-6 present in the human's genome.

73. (NEW) The method of claim 71 wherein the polymorphic form is a single nucleotide polymorphism (SNP).